Paper No. 18

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Ex parte DAVID N. HERNDON

Appeal No. 2004-2170
Application No. 09/901,429

ON BRIEF

Before SCHEINER, ADAMS and GRIMES, Administrative Patent Judges.

SCHEINER, Administrative Patent Judge.

DECISION ON APPEAL

This is a decision on appeal under 35 U.S.C. § 134 from the final rejection of claims 1-7 and 9-12. Claims 14-20 are also pending, but have been withdrawn from further consideration by the examiner.

Claims 1, 3, 4 and 9 are representative of the subject matter on appeal:

1. A method of treating an individual having a severe burn, comprising the step of administering to said individual a pharmacologically effective dose of a beta-adrenergic antagonist, wherein treatment with said beta-adrenergic antagonist improves skeletal muscle protein kinetics in said individual as compared to [an] individual without said treatment.

3. The method of claim 2, wherein said beta-adrenergic antagonist is administered in a dose that decrease[s] heart rate in said individual by about 25%.

4. The method of claim 2, wherein said beta-adrenergic antagonist is administered in a dose of from about 0.1 mg/kg of the body weight of the individual to about 10 mg/kg of the body weight of the individual.

9. A method of treating an individual having a severe burn, comprising the step of administering to said individual a pharmacologically effective dose of propranolol,

wherein treatment with said propranolol improves skeletal muscle protein kinetics in said individual as compared to [an] individual without said treatment.

The reference relied on by the examiner is:

Herndon et al. (Herndon), "Lipolysis in Burned Patients is Stimulated by the β_2-Receptor for Catecholamines," Arch. Surg., Vol. 129, pp. 1301-1305 (December 1994)

Claims 1-7 and 9-12 stand rejected under 35 U.S.C. § 102(b) as anticipated by Herndon. We reverse.

BACKGROUND

> The hypermetabolic response to severe burn is associated with increased energy expenditure and substrate release from protein and fat stores. After severe trauma, net protein catabolism is increased which leads to loss of lean body mass and muscle wasting. Muscle proteolysis continues for at least 9 months after severe burn which predisposes patients to delays in rehabilitation, and increased morbidity and mortality.
>
> Endogenous catecholamines are primary mediators of the hypermetabolic response to trauma or burn. Shortly after severe trauma or burn, plasma catecholamine levels increase as much as 10-fold. This systemic response to injury is characterized by development of a hyperdynamic circulation, elevated basal energy expenditure, and net skeletal muscle protein catabolism.

Specification, page 2 (citations omitted).

> The present invention demonstrates that blockade of β-adrenergic stimulation with orally administered propranolol decreases resting energy expenditure and net muscle catabolism. Twenty-five [ ] severely burned . . . children were studied . . . Thirteen of the subjects received oral propranolol for at least two weeks, and twelve served as non-treated controls. Propranolol was titrated to decrease resting heart rate 20% from the patient's baseline. Resting energy expenditure [ ] and skeletal muscle protein kinetics were measured before and after two weeks of β-blockade . . .
>
> During beta blockade, heart rates and resting energy expenditures of the propranolol group were lower than baseline and lower than those of the control group ($p<0.05$). Corresponding to the significant differences in heart rate and resting energy expenditure, muscle protein net balance improved 82% relative to pre-treated baseline in the subjects treated with propranolol while it decreased 27% in the non-treated control subjects ($p<0.05$) . . .

Specification, pages 3-4.

Finally, the specification indicates that "[p]ost-traumatic net proteolysis is primarily a result of a large increase in protein degradation, which outweighs a lesser increase in total protein synthesis" (id., page 30), but "[p]ropranolol induced an increase in the intracellular recycling of free amino acids" (id.), and "[a]n acceleration in protein synthesis in propranolol treated subjects was seen" (id.). When patients were tested after four weeks of treatment with propranolol, "[t]he net balance of protein synthesis and breakdown [had] achieved anabolic levels" (id., page 29).

DISCUSSION

Independent claim 1 is directed to a method of treating a burn patient by administering a pharmacologically effective dose of a beta-adrenergic antagonist, wherein treatment with the beta-adrenergic antagonist improves skeletal muscle protein kinetics in the patient as compared with an untreated patient. Dependent claim 3 specifies that the dose is effective to decrease the patient's heart rate by about 25%.

There is no dispute that Herndon administers propranolol, a β-adrenergic antagonist, to burn patients, at a dose of 2 mg/kg per day – a dose effective to decrease patients' heart rates to the required level, and also within the range asserted in the specification to be effective in improving skeletal muscle protein kinetics (see pages 7-8). According to the examiner, Herndon anticipates the claimed invention because "the critical elements (i.e., the effective therapeutic dosage regimen (2 mg/kg), the burn patients and the successful outcome) required by the instant claims have been taught and acknowledged" (Answer, page 4). As further explained by the examiner, "the underlying mechanism recited in the claims (i.e., [skeletal] [muscle] protein kinetics) [is] not considered as a critical element having [patentable] weight because the

outcome of the treatment is the same regardless . . . Since [the] same therapeutic modality [is] taught and the same patient is also used in the treatment, there is no difference between the claimed subject matter and the conventional treatment" (id., page 5).

On the surface, the examiner's position has merit. On cursory review, Herndon does appear to describe all of the manipulative steps required by the claims, and it is well established that merely discovering and claiming a new benefit of an old process cannot render the process again patentable. See In re Woodruff, 919 F.2d 1575, 1577, 16 USPQ2d 1934, 1936 (Fed. Cir. 1990). Thus, the mere fact that "Herndon [ ] did not teach or suggest [that] treatment with β-adrenergic antagonists had an inherent feature of improving skeletal muscle protein kinetics" (Brief, page 9) would not "render the process again patentable."

Nevertheless, we find that the examiner has not established that Herndon's method anticipates all of "the critical elements" (Answer, page 4) of the claims. As always, "[a]nalysis begins with a key legal question – what is the invention claimed?" since "[c]laim interpretation . . . will normally control the remainder of the decisional process," Panduit Corp. v. Dennison Mfg. Co., 810 F.2d 1561, 1567-68, 1 USPQ2d 1593, 1597 (Fed. Cir.), cert. denied, 481 U.S. 1052 (1987).

To meet the requirements of the claims on appeal, a "pharmacologically effective dose" of a beta-adrenergic antagonist must be a dose effective to improve skeletal muscle protein kinetics in a burn patient, as compared with an untreated patient. According to the present specification, a beta-adrenergic antagonist (propranolol) administered for two weeks, "improved muscle protein net balance from baseline [ ] and as compared with non-treated controls" (specification, page 23). After four weeks of treatment, "[a]n acceleration in protein synthesis in propranolol treated subjects was

seen" (id., page 30), and "[t]he net balance of protein synthesis and breakdown achieved anabolic levels" (id., page 29), while "[f]at-free mass, corresponding to the sum of lean mass and bone mass . . . was preserved . . . In comparison, ten untreated time control subjects lost 9% of their fat-free mass . . ." (id.). "In summary," according to the specification, "long term β blockade decreases lean mass catabolism in severely burned children" (id., page 32). Thus, to meet every element of the claimed invention, propranolol treatment must have a positive effect on muscle protein net balance as compared with non-treated controls.

On the other hand, Herndon administered propranolol at an initial dosage of 2 mg/kg body weight per day, for five days, and observed a decrease in heart rate and lipolysis, but "failed to document an effect of propranolol . . . on protein kinetics[,]" even though "two independent approaches for assessing net protein breakdown" were used (Herndon, page 1304). It may be that a five day course of propranolol was not long enough to improve protein kinetics, or it may simply be that there were other differences in methodology that resulted in Herndon's failure to document an effect on protein kinetics. In any case, the examiner has not explained how Herndon's finding that propranolol had no effect on protein kinetics can be consistent with the examiner's assertion that Herndon's method results in an improvement in skeletal muscle protein kinetics.

On this record, the examiner has not established that Herndon anticipates every element of the claims on appeal. The rejection of claims 1-7 and 9-12 is reversed.

REVERSED

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